Alerts, Notices, and Case Reports

Illness Associated With Eating Seaweed, Hawaii, 1994

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SEVEN OF 13 PEOPLE (54%) at a Labor Day picnic on the island of Hawaii in 1994 became ill with gastrointestinal symptoms within 15 to 90 minutes of eating lunch. We conducted a retrospective cohort study to determine what food served at the picnic was related to the illness. All 7 people who ate the seaweed preparation became ill. Two of them ate less than one serving (defined as one serving spoon) of seaweed and experienced only a burning sensation in the mouth and throat; the 5 who ate one or more servings experienced more serious gastrointestinal illness. The seaweed was identified as Gracilaria coronopifolia, a species usually considered to be nontoxic. Water sample analysis from the harvest area indicated an increased level of enterococci in the water during the month of harvest, although the increase was not above acceptable standards. Mouse and guinea pig assays revealed that the seaweed contained debromoaplysiatoxin and aplysiatoxin, two toxins that possibly came from the blue-green algae found on the seaweed. This is the first reported outbreak of seaweed poisoning in Hawaii; in Guam, a previously undescribed toxin in Gracilaria tsudai caused the death of 3 people in 1991. Data from both incidents suggest that seaweeds usually considered nontoxic may occasionally produce poisons associated with illness in humans.

In the Pacific Islands, seaweed (algae) is often served as a side dish at meals, and it is an important component in the diet of many who reside in the Pacific Rim. The seaweed is usually obtained by harvesting it at beaches, gathering it in near-shore waters, or purchasing it at local markets. It is served either raw or cooked and is often eaten with salt or mixed with spices such as garlic, chili pepper, soy sauce, and ginger. Although illness after consumption of seaweed has been reported in other parts of the Pacific (Guam, 1 California, 2 and Japan 3), seaweed toxicity has never been reported to the Hawaii Department of Health.

On Sept. 9, 1994, a 60-year-old woman on the island of Maui telephoned the local health department to report a burning sensation in her mouth and throat that began 15 minutes

(Marshall KLE, Vogt RL. Illness associated with eating seaweed, Hawaii, 1994. West J Med 1998; 169:293–295) after she had tasted a seaweed or *ogo* preparation. The woman had washed the seaweed, boiled it in water for 1–2 minutes, and then mixed it with vinegar, sugar, soy sauce, and sliced cucumbers. Six hours after tasting the dish, she became ill with a headache and generalized myalgia. When examined by a physician the next day, she had a headache and a sore throat and mouth. A throat culture was negative for group A streptococcus. No other household members had tasted the seaweed, and samples were unavailable because the woman had disposed of the seaweed after becoming ill. She had received the seaweed on Sept. 1, 1994, from her son's friend, who had gathered the seaweed at a site in a bay on a northeast-facing windward shore in Maui County.

Six days earlier, on Sept. 3, 1994, a 49-year-old woman on the island of Hawaii had received seaweed obtained from the same source. When later contacted by the local district health department, she reported a burning sensation in her mouth and throat that lasted about 4 hours after tasting the preparation she had made from the seaweed. The woman's 50-year-old husband had also tasted the preparation and reported a similar burning sensation. At the time, both had attributed the sensation to chili peppers.

The seaweed was refrigerated until it was prepared on September 4. The woman cleaned it; boiled it for 3–4 minutes; rinsed it; mixed it with codfish, vinegar, onions, shoyu, chili peppers, and tomatoes; and served the preparation at a Labor Day picnic the following day. The local health department contacted those who had attended the picnic and found that 7 of the 13 attendees had gastrointestinal illness characterized by nausea and diarrhea soon after eating at the picnic. This report summarizes the epidemiologic investigation of those illnesses and discusses several other seaweed-associated outbreaks in recent years.

Methods

The Hawaii Department of Health conducted a retrospective cohort study of all picnic attendees to determine whether illness was caused by eating the seaweed served at the picnic. A survey questionnaire was administered by telephone; picnic attendees were asked which foods they had consumed, if and when onset of illness had occurred, and symptoms of illness. A case was defined as illness in a picnic attendee who, within 2 hours of eating food items from the picnic, had either a burning sensation in the mouth or throat or two or more of the following symptoms: vomiting, diarrhea, nausea, and lethargy. Food-specific illness rates were calculated using Epi-Info 6.01.

Both the seaweed served at the picnic and another sample obtained from the same harvest site were examined at the University of Hawaii for species identification and toxicity studies. The algae were rinsed well and extracted with acetone. The toxicity of the crude extract was checked by intraperitoneal injection into mice and observation of the mice for symptoms of toxicity. High-performance liquid

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	Rainfall (past 24 h)			
Month	North Point	South Point	North Point	South Point
July	No	No	1	1
August	No	No	1	1
September	No	No	4	50
October	Yes	Yes	1	1
November	*	_*	1	1
December	No	No	1	1

chromatography was used to isolate the exact compound that caused the mice to exhibit the most extreme signs of toxicity. Bacterial organisms on the surface and within the algae were identified microscopically. The bacteria were cultured using 2% NaCl heart infusion agar (DIFCO). Each culture was then extracted, and the extracts were subjected to the mouse toxicity test.⁴

Results

Eight adults and 5 children attended the picnic. Seven (54%) of the 13 attendees became ill within 15–90 minutes of eating the meal, all adults (5 men and 2 women). Of those, 5 reported diarrhea, 5 nausea, 3 vomiting, and 4 a burning sensation. Gastrointestinal symptoms lasted a mean of 22 hours. None of those who fell ill sought medical treatment.

Menu items for the picnic included poi, two types of fish (mamo and awe), beef stew, the seaweed preparation, salmon salad, steak, rice, tuna salad, chips, crackers, beer, soda, and water. Only the 7 who had eaten the seaweed preparation became ill (RR undefined, P < 0.05). Of 8 people who ate the salmon salad, the only other food item besides seaweed that was significantly associated with illness, 7 became ill (RR undefined). The seaweed, however, was the only food item that had been eaten by all the ill persons and had not been eaten by those who remained well. Two (29%) of the 7 ate less than one serving (defined as one serving spoon) of seaweed and experienced only a burning sensation in the mouth and throat, whereas the 5 who ate one or more servings had gastrointestinal illness as well.

The implicated seaweed had been harvested from a bay into which two fresh water streams flow at the northern and southern points; in addition, storm drain discharge from the city flows into the stream at the southern point of the bay, and the tides transport tree branches and other debris into the bay. The Hawaii Department of Health's State Wastewater Management Division, Clean Water Branch, collects water samples from this area on a monthly basis and measures rainfall during the 24-hour period before sample collection. The samples are tested for enterococci levels, and any levels <70 colonies/100 ml are considered acceptable by Hawaii Administrative Rules.⁵

The Hawaii Department of Health records of water samples tested during July through December 1994 indicated no unacceptably elevated levels of enterococci (Table 1). Enterococci levels in all those months but one were 1 colony/100 mL; in September, the samples collected from stream mouths at the northern and southern points of the bay (about 1.5 miles apart) were 4 and 50 colonies/100 ml, respectively. According to the Hawaii Department of Health, the current usually flows from south to north, and the bay is most affected by the outflow at the southern end of the bay. No sewage spills, pesticide contamination, or other potential pollutants were reported in the area in the period of time before the seaweed was harvested.

The seaweed was identified as *Gracilaria coronopifolia* with blue-green algae attached and interwoven among the stems of seaweed. Colonies of two different species of *Pseudomonas* and *Vibrio* grew on culture media. Mice injected with extracts from the bacteria exhibited no signs of toxicity other than transient weakness. Extracts from the seaweed caused symptoms of toxicity in the mice; such symptoms ranged from diarrhea at low doses to death within 15 minutes after injection of the most toxic isolate. Two toxic compounds, identified as debromoaplysiatoxin and aplysiatoxin, were isolated using high-performance liquid chromatography.⁴

Discussion

Results of the epidemiologic study implicating seaweed as the source of illness were supported by a doseeffect relationship: those who had eaten only a taste of the seaweed had less severe symptoms than those who had eaten one or more servings.

Laboratory findings of slightly increased levels of enterococci in the seawater at the time the seaweed had been harvested indicated potential microbiologic contamination of the water, even though the levels were within the acceptable water quality standards established by the Hawaii Department of Health. Enterococci testing of water samples does not differentiate between human or animal fecal enterococci and nonpathogenic soil enterococci, and no other causative biologic agents were found in the water. Bacterial organisms were found on and in the algae, but extracts from the bacteria did not cause a toxic reaction when injected into mice. Additional laboratory studies isolated two toxins from the seaweed mix, debromoaplysiatoxin and aplysiatoxin; those toxins caused similar illness in mice and killed them at higher doses.

Blue-green algae was found on the *G. coronopifolia*. Debromoaplysiatoxin and aplysiatoxin are usually found in blue-green algae, specifically the *Lyngbya majuscula* in Hawaii. Although freshwater cyanobacteria have been associated with outbreaks of illness, this is the first gastrointestinal outbreak known to be associated with debromoaplysiatoxin and aplysiatoxin in marine blue-green algae. Aplysiatoxin and debromoaplysiatoxin can cause skin irritation characterized by redness, blisters, and a burning or itching sensation of the skin^{8,9}; both have been implicated in occurrences of chemical "swimmer's itch" on

various north shore beaches of Oahu. Both are also known to be potent tumor promoters in two-stage carcinogenesis in mouse skin.¹⁰ The potential of these two toxins as tumor promoters in humans is unknown; but their noxious effects are a public health concern. Swimmers and seaweed on leeward shores of the Hawaiian islands rarely come in contact with blue-green algae because trade winds usually transport the algae away from the shore. Slightly elevated enterococci levels in seawater might serve as a signal for a potential health threat of harvesting seaweed for consumption.

Environmental stresses on the seaweed possibly allowed for colonization of the blue-green algae. Previous studies have demonstrated that stress from over-harvesting might cause seaweed to produce toxins as a method of protection and that environmental changes and increased pollution promote colonization of toxin-producing bacteria.¹¹ Although unlikely in this case, other studies have suggested that some species of seaweed may become toxic at the end of their reproductive cycle, and therefore exhibit a seasonal variation in toxin production. 12

Although this was the first reported case of seaweedinduced illness in Hawaii, several cases have been reported in the Pacific Rim.^{3,13} In 1991, 13 people became ill eating seaweed (Gracilaria tsudai) harvested in Guam¹: 3 died. In addition to gastrointestinal illness, they experienced fever, wheezing, muscle fasciculations, and hypotension. In 1992, according to a press report, 3 people became ill, one of whom was hospitalized, after eating seaweed (Grasilariopsis lemanaeformis) picked on the beach of Half Moon Bay in California.2 In 1993, 2 people became ill, 1 of whom later died, after eating Gracilaria verrucosa seaweed in Japan.³ In the cases in Guam and California, the implicated seaweed was not previously known to be toxic. In the two cases that occurred in Japan, the seaweed was not examined adequately and no toxin was identified. Whether the dearth of reported seaweed-associated illness reflects failure to report illness or infrequent occurrence is unclear.

Since the incident in Hawaii, physicians were alerted to the potential for seaweed-induced toxicity and were requested to report cases to the Hawaii Department of Health. Seaweed has been collected from the same harvest site for toxin surveillance. Until further information regarding seaweed toxicity is known, those who harvest seaweed for consumption should be aware that any seaweed harvested from Hawaiian waters is potentially toxic. Those who experience a burning sensation after eating a seaweed preparation should notify the Hawaii Department of Health.

Although the species of blue-green algae was not identified in this outbreak, Lyngbya majuscula, which possibly contains debromoaplysiatoxin and aplysiatoxin and can grow epiphytically on certain edible seaweeds,8 has been identified in Hawaii.9 The Hawaii Department of Health recommends that seaweed to which blue-green algaespecifically Lyngbya species—is attached should not be eaten. Boiling the seaweed did not inactivate the toxin in the two incidents on Maui and Hawaii. Because the consumption of seaweed is common among many ethnic groups who reside in the Pacific area, additional research is

necessary to determine the etiology and extent of seaweedassociated illnesses and to characterize such illnesses.

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Sphenoid Pneumoceles Cause Episodic Pressure-Related Blindness

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THOUGH ETIOLOGIES VARY, transient visual loss in adults is most commonly caused by vascular insufficiency secondary

(Hanasono MM, Norbash AM, Shepard K, Terris DJ. Sphenoid pneumoceles cause episodic pressure-related blindness. West J Med 1998; 169:295-299)

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